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EXHIBIT A

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CAREER SUMMARY

Four years experience in biopharmaceutical drug discovery, developing cell-based assay for identifying small molecule drug candidates and establishing transgenic animal models for studying angiogenesis and inflammation mediated diseases. All inclusive, 13 years experience in molecular and cell biology and animal modeling, expertise in molecular techniques, angiogenesis and inflammation transgenic mice models and luciferase "knock-in" mice production, protein purification, eukaryotic gene expression, bacterial genetics.

Initiation of drug discovery program at Tularik Corp., identifying novel targets for drug discovery, conducting proof-of-concept studies, designing and implementing assays.

Postdoctoral work at Queensland Institute of Medical Research, cloned ATM gene and demonstrated that wild type ATM gene expression in lymphablasts from patients of Ataxia-Telangiectasia (A-T) syndrome can correct radiation sensitive phenotype of the A-T cells.

Postdoctoral work at Griffith University, purified L-asparaginase from guinea pig serum.

Published 12 papers in peer reviewed journals including Proc. Natl. Acad. of Sci. and Nature. Contributed a protocol chapter to the lab manual "Current Protocol in Molecular Biology". Issued six U.S. patents on mitochondrial uncoupling protein research, filed two patent applications on creating angiogenesis transgenic mice model for monitoring tumor development, wound healing and screening anti-angiogenesis compounds.

EDUCATION BACKGROUND

Ph.D Microbial Molecular Genetics, University of Adelaide, Australia Oct. 1993
Conducted molecular and genetic analysis of a plasmid from anearobic bacterial strain *Selenomonas* and developed a series of shuttle vectors.

B.S. Animal Science, Lai Young Agricultural University, China July 1986

PROFESSIONAL EXPERIENCE

Xenogen Corporation, Alameda, CA 94501
Project Leader, *Senior Research Scientist* April 1999-present
Research topic: Creating angiogenesis murine models for human tumor development

My current research at Xenogen Corporation aims to creating angiogenesis animal models for studying aniogenesis related human diseases such as tumor development and cardivascular disorders. For example, angiogenesis is a critical step of tumor development. Most of the human tumors can not grow more than 2-3 mm in diameter without microvessel formation inside the tumor, an angiogenic process which provides the tumor with all the necessary nutrients through blood circulation. Tumor angiogenic

processes are triggered by tumor secreted vascular endothelial cell growth factor (VEGF). This factor binds to several endothelial cell growth factor receptors, including VEGFR2, that are located on the surface of endothelial cells and promote endothelial cell proliferation and blood vessel formation. Other angiogenic factors, such as tyrosine kinase receptor Tie2, are also involved in tumor angiogenic processes. All these angiogenic factors are highly expressed in the majority of primary human tumors. The expression level of these angiogenic factors determines how rapidly the angiogenic processes will preceed. At Xenogen Corporation, I have been focused on creating transgenic mice in which the promoters of VEGF, VEGFR2 and Tie2 are used to drive the expression of the luciferase gene. The level of luciferase expression at each stage of the angiogenesis process can be directly monitored in live transgenic mice using an intensified CCD camera system, thus provides valuable information on how the expression of angiogenic factors contributes to tumor development and metastasis. The murine angiogenesis model can be used for mid to high-through screening of novel compounds that can inhibit the expression of the angiogenic factors in vivo, which may lead to the discovery of new anti-tumor drugs.

POST-DOCTORAL RESEARCH

Tularik Inc., South San Francisco, CA 94080

Obesity/Diabetes Group, Laboratory of Dr. Jin-Long Chen May 1997-Mar.1999

Research topic: Gene regulation study and development of cell based assay for UCP2 and UCP3

At Tularik, I worked on two members of the mitochondria uncoupling protein family, UCP2 and UCP3. UCPs are involved in thermogenesis through uncoupling cellular energy transmission processes. During the metabolic process, a significant proportion of the metabolic energy is dissipated as heat instead of being converted to ATP due to the uncoupling activity of the UCPs. It is widely accepted that UCPs are critical to body energy homeostasis and that increasing the expression of UCPs could be a novel approach to treating obesity. In 1997, I became the first one to discover mouse UCP3 gene (U.S. Pat No. 5 846 779). The expression of this gene is restricted to skeletal muscle and is up-regulated by T3 thyroid hormone, beta-adrenoceptor agonist and cold exposure. The discovery that UCP3 is physiologically regulated lead to the initiation of an anti-obesity drug discovery program at Tularik. I successfully created UCP3 promoter-luciferase "knock-in" mice and showed that luciferase expression is skeletal muscle specific. thus providing a perfect animal model for studying UCP3 gene regulation. Subsequently, a luciferase expressing myoblast cell line was developed from these mice and used by the drug discovery group at Tularik in a high throughput screen for compounds that up-regulate the UCP3 gene. Using a similar approach, I also created UCP2 promoter-luciferase "knock-in" mice and showed, as expected, that luciferase is predominantly expressed in adipose tissue. An adipocyte cell line was developed from the UCP2-luciferase "knock-in" mice and a high throughput screen for compounds that up-regulate the UCP2 gene is ongoing. Apart from my involvement in the initiation of the anti-obesity drug discovery program, I also contributed to the cloning and analysis of UCP3 promoter (U.S. Pat No. T5 849 581) and initiated additional transgenic mice programs to investigate whether UCP2 and UCP3 over-expression can cause weight loss.

Accomplishments Summary:

- Cloned mouse UCP3 cDNA and carried out in vivo gene regulation studies
- Cloned and functionally analyzed mouse UCP3 promoter
- Created Ap2 promoter-hUCP2 and MCK promoter-hUCP3 constructs for over-expressing UCP2 and UCP3 genes in transgenic mice in a tissue specific manner

- Created UCP3-luciferase and UCP2-luciferase chimeric mice using targeted "knock-in" technology
- Developed UCP2 and UCP3 cell based assays for high throughput screening of anti-obesity compounds
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Queensland Institute of Medical Research, Brisbane, Australia

Laboratory of Professor Martin Lavin Feb.1995-April 1997

Research topic: Cloning and functional studies of the ATM gene

Accomplishments:

Ataxia-Telangiectasia (A-T) is a rare hereditary syndrom involving cerebellar degeneration, immunodeficiency, cancer risk, and radiosensitivity. This genetic defect is caused by a single mutation of a gene named ATM. In mid 1995, a group led by Dr. Shilo cloned a partial cDNA of the ATM gene, which sparkled a world wide interest to clone the full length ATM cDNA for functional studies of its encoded product. However, several labs around the world failed in cloning of the full length ATM cDNA due to its large size (close to 10 kb) and instability in *E.coli*. I overcomed all these problems and became the first one that cloned full length ATM cDNA. Subsequently, in colaboration with others, I demonstrated that wild type ATM protein expression in A-T cells can correct the radiosensitive phenotypes, rendering the A-T cells more resistant to gama-radiation induced DNA damage. This work provided critical basis for future gene therapy to fight against A-T. My other research activities includes the demonstration that anti-cancer drug cisplatin can drive A-T cells to apoptosis by increasing the cellular level of p53 protein. In collaboation with my collegues, I contributed to the discovery of DNA-dependent protein kinase as a key death substrate in apoptosis.

Accomplishments Summary:

- Cloned the full length human ATM cDNA and studied ATM gene functions in stably transfected A-T cells with ATM expression vectors
- Conducted research on ATM mediated transcriptional regulation of p53 in Ataxia-Telangiectasia (A-T) lymphoblasts treated with DNA damaging agents
- Achieved recombinant ATM protein expression using baculovirus protein expression system
- Contributed to the discovery of DNA-PK as a key death substrate in apoptosis
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Griffith University, Brisbane, Australia

Laboratory of Professor Ifor Beacham Oct.1992-Jan.1995

Research topic: Guinea pig asparaginase protein purification

Accomplishments

- Purified L-asparaginase from guinea pig serum and produced L-asparaginase specific polyclonal antibodies

GRADUATE STUDIES:

University of Adelaide, Australia

Department of Animal Sciences, Laboratory of Dr. John Brooker July.1988-Oct..1992

Research topic: Molecular genetic study of anearobic bacterial species *Selenomonas ruminantium*

Ph.D Thesis: Ning Zhang (1993) Molecular characterization of ruminal bacterial species Selenomonas ruminantium

HONORS AND AWARDS:

- NHMRC (Australia) large grant (1997-2002) for ATM research

- Chinese Education Committee Scholarship for outstanding students (1988-1992)

PATENTS ISSUED:

Zhang, N., Amaral C., and Chen, J-L., 2001. UCP3 genes. (Pat No. 6 248 561)

Zhang, N., Amaral C., and Chen, J-L., 2000. Murine UCP3 polypeptides. (Pat No. 6 025 469)

Zhang, N., Amaral C., and Chen, J-L., 1998. Nucleic acids encoding murine UCP3 genes. (Pat No. 5 846 779)

Zhang, N., Amaral C., and Chen, J-L., 1999. Murine UCP3 polypeptides. (Pat No. 5 952 469)

Amaral C., **Zhang, N.** and Chen, J-L., 1998. Regulators of UCP3 gene expression. (Pat No. 5 849 581)

Amaral C., **Zhang, N.** and Chen, J-L., 1999. Assays for identifying agents which affect regulators of UCP3 gene expression. (Pat No. 5 976 808)

INVITED ARTICLE

Zhang, N. and Chen, J-L.,1998 Purification of recombinant proteins and study of protein interaction by epitope tagging. p10.15.1-10.15.9, In F.M. Ausubel, R. Brent, R.E. Kingston, D.D. Moore, J.G. Seidman, J.A. Smith and K. Struhl (Eds) Current Protocol in Molecular Biology, John Wiley & Sons, New York

RESEARCH PAPERS

Zhang, N., Chen, P., Khanna, K.K., Scott, S., Gatei, M., Kozlov, S., Watters, D., Spring, K., Yen, T., Lavin, M.F., (1997) Isolation of full-length ATM cDNA and correction of the ataxia-telangiectasia cellular phenotype. ***Proc Natl Acad Sci*** 94:8021-8026

Shafman, T, Khanna, K.K., Kedar, P., Spring, K., Kozlov, S., Yen, T., Hobson, K., Gatei, M., **Zhang, N.**, Watters, D., Egerton, M., Shiloh, Y., Kharbanda, S., Kufe, D., Lavin, M.F., (1997) Interaction between ATM protein and c-Abl in response to DNA damage. ***Nature*** 387:520-523

Watters D, Khanna KK, Beamish H, Birrell G, Spring K, Kedar P, Gatei M, Stenzel D, Hobson K, Kozlov S, **Zhang N**, Farrell A, Ramsay J, Gatti R, Lavin M. 1997 Cellular localisation of the ataxia-telangiectasia (ATM) gene product and discrimination between mutated and normal forms. ***Oncogene*** 14:1911-1921

Zhang N, Chen, P., Gatei, M., Scott, S., Khanna, K.K., Lavin, M.F., (1998), An anti-sense construct of full length ATM cDNA imposes a radiosensitive phenotype on normal cells. ***Oncogene*** 17:811-8.

Liao WC, Haimovitz-Friedman A, Persaud RS, McLoughlin M, Ehleiter D, **Zhang N**, Gatei M, Lavin M, Kolesnick R, Fuks Z, Ataxia Telangiectasia-mutated Gene Product Inhibits DNA Damage-induced Apoptosis via Ceramide Synthase. **J Biol Chem** 1999 274:17908-17917

Scott, S.P., **Zhang, N.**, Khanna, K.K., Khromykh, A., Hobson, K., Watters, D., Lavin, M.F., (1998) Cloning and Expression of the Ataxia-Telangiectasia Gene in Baculovirus. ***Biochem Biophys Res Commun*** 245:144-148

Song Q, Lu H, Zhang N, Luckow B, Shah G, Poirier G, Lavin M Specific cleavage of the large subunit of replication factor C in apoptosis is mediated by CPP32-like protease. Biochem Biophys Res Commun 1997 233:343-8

Song, Q. Z., Lees-Miller, S.P., Kumar, S., **Zhang, N.**, Chan, D.W., Smith, G.C.M., Jackson, S.P., Alnemri, E.S., Litwack, G. and Lavin, M.F., (1996) DNA-dependent protein kinase catalytic subunit: A target for an ICE-like proteinase in apoptosis. ***EMBO J.*** 15:3238-3246.

Zhang, N., Song, Q.Z., Lu, H., and Lavin, M.F., (1996) Induction of p53 and increased sensitivity to cisplatin in Ataxia-Telangiectasia. ***Oncogene*** 13:655-659.

Zhang, N., Clarke, F., Di Trapani, G., Keough, D., and Beacham, I.R., (1995) Guinea pig serum L-asparaginase purification, and immunological relationship to liver L-asparaginase and serum L-asparaginases in other mammals. ***Comp. Biochem. Physiol.*** 112B 607-612.

Zhang, N., Brooker, JD., (1993) Characterization, sequence, and replication of a small cryptic plasmid from *Selenomonas ruminantium* subspecies *lactilytica*. ***Plasmid*** 1993 29:125-134

Zhang, N., Attwood, G.T., Lockington, R.A. and Brooker, J.D., (1991) Genetic diversity in ruminal isolates of *Selenomonas ruminantium*. ***Current Microbiology*** 22: 279-284.

PUBLISHED ABSTRACTS:

1. Brooker, J.D., Miller, S., **Zhang, N.** and Stokes, B. (1989) Gene and monoclonal antibody probes for rumen bacterial analysis. *Proc. Aust. Soc. for Microbiology.* 10: 330

2. Brooker, J.D., **Zhang, N.**, & Clarke, K. (1990) Measurements of microbial diversity in the rumen. *Proc. Aust. Soc. for Microbiology.* 11

3. **Zhang N.**, Lockington R.A., and Brooker, J.D. (1991) Genetic diversity in ruminal isolates of *Selenomonas ruminantium*. *Proc. Aust. Soc. for Microbiology.* 12: 257.

4. **Zhang N**, Li B., Weber A., Madlansacay M.R., Steinmetz K.L., Purchio A.F., 2001 Whole Body Imaging of Vascular Endothelial Growth Factor Receptor-2 Expression Patterns in Transgenic Mice Abstract ID: 867

5. Li B., **Zhang N**, Weber A., Lyons R., Madlansacay M.R., Steinmetz K.L., Purchio A.F., 2001 Whole body Imaging of inducible nitric oxide synthase expression patterns in transgenic mice treated with lipopolysacchride and interferon-gamma. 2001 Society of Toxicology, 40th Annual Meeting, San Francisco, CA Abstract ID: 839

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6. **Zhang N**, Li B., Weber A., Sambucetti L.C., Purchio A.F., 2001 In Vivo Monitoring of VEGF Expression in Primary and Metastatic Tumors with the IVISTM Imaging System 2001 American Association for Cancer Research, New Orleans, LA